# Improved Motor Activation of C7 Myotome in Previous Incomplete Cervical Spinal Cord Injury via Spinal Cord Stimulation

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Title:

**Improved Motor Activation of C7 Myotome in Previous Incomplete Cervical Spinal Cord Injury via Spinal Cord Stimulation**

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*KEY CLINICAL MESSAGE*

*The sequela of spinal cord injury (SCI) affects millions worldwide. We present a case of a 60-year-old male with a SCI, who experienced functional improvement and a significant reduction in pain following spinal cord stimulation (SCS) treatment. This promising outcome suggests SCS could be a future cornerstone in SCI management.*

## INTRODUCTION

Spinal cord injury (SCI) is a serious medical condition that leads to significant morbidity and mortality. In the United States alone, there are approximately 300,000 people living with SCI, and about 17,000 new cases are reported each year with a cost of almost $10 billion annually[1]. Approximately two-thirds of SCIs are incomplete injuries, where some neural activity still remains below the lesion, thus preserving partial function. Unfortunately, studies have reported that even in patients with incomplete SCI, there is a substantial decrease in quality of life following their injury, along with a significant reduction in life expectancy [2,3].

The outcome of a SCI varies depending on the location and degree of neurological damage. Some complications, such as the motor spasticity seen in our patient, affects 65–92% of people with chronic SCI and is more common with higher levels of injuries [3,4,5]. Spasticity is a velocity-dependent increase in muscle tone associated with upper motor neuron (UMN) injuries [6]. The damaged UMN leads to a loss of inhibitory signals in the descending spinal tracts, resulting in increased stretch reflex and muscle tone[7]. Spasticity typically begins to develop several weeks or months after injury as the period of areflexia begins to subside [8] and can lead to pain, discomfort, and complications with significant functional impairment, further contributing to decreased quality of life for patients[6, 2].

Epidural spinal cord stimulation (SCS) is a neuromodulation technique that places electrodes within the epidural space of the dorsal column to deliver mild electrical impulses. Although SCS has traditionally been employed as a treatment for chronic pain, recent research suggests its potential usefulness for other medical conditions as well. A 2022 systematic review found that SCS could be beneficial in restoring sensorimotor function, including volitional movement, after SCI. The total participants included 327 patients with SCI, and of the studies assessing sensorimotor function, 71/127 (56%) of patients regained volitional movement during SCS [9]. In addition, a 2024 systematic review analyzed thirty-four studies for spasticity improvements with the use of SCS. A subset of their data looked specifically at subjective improvement in spasticity after spinal cord injury, where 190/281 (68%) of patients found improvement in their symptoms after SCS [10]. This data shows that neuromodulation holds promise as a tool that may enhance patients' functional recovery in conditions beyond pain such as SCI and assist activity-based recovery. In this case report, we hope to add

to this potential by presenting a patient who had a long-standing history of pain and spasticity as a result of SCI, and found impactful improvements with the use of spinal cord stimulation.

## CASE HISTORY

A 60-year-old male with a history of traumatic cervical SCI secondary to a ski accident presented to the pain management clinic with chronic spasticity and neuropathic pain of the right upper extremity (RUE) and bilateral lower extremities (BLE). The patient had previously found minimal relief with conservative management and chemodenervation with botulinum toxin. Physical examination demonstrated increased tone in his RUE, resulting in loss of function, along with complaints of paresthesia and numbness in his BLE. After thorough testing according to the International Standards for Neurological Classification of SCI (ISNCSCI), the patient's injury was classified as C5 AIS D.

## METHODS

The patient was agreeable to trying other treatment modalities and subsequently underwent a spinal cord stimulation (SCS) trial, with cervical and thoracic leads placed to cover mid-C3-C5 and mid-T9-T11, respectively. This trial revealed a significant improvement in the patient's right-sided neuropathic pain, demonstrating a greater than 70% reduction in symptoms. This positive outcome supported the continuation of the current treatment strategy, leading to the scheduled implantation of a SCS.

During the SCS implant, two 8-contact spinal cord stimulator leads were inserted and advanced under AP and lateral fluoroscopic guidance into the posterior epidural space. The left lead was advanced to the level of C5, and the right lead to the level of C4 (Fig. 1). The thoracic leads were placed midline at the level of the T11 vertebral body (Fig. 2). The placement of the leads within the posterior epidural space was confirmed with lateral fluoroscopy (Fig. 3). Intraoperative paresthesia stimulation confirmed adequate coverage of the pain areas, and the leads were anchored and battery placed in a typical fashion. The patient was awake and conversant throughout the procedure.

## CONCLUSION AND RESULTS

Two weeks post-procedure, the patient reported improvement in his hand spasticity and neuropathic pain. Neuropathic pain in the feet was mostly masked by the stimulator. However, he was still able to notice it and adjust settings for better coverage. Subsequent weeks involved several programming adjustments to optimize pain management and spasticity reduction. These adjustments led to the restoration of the patient's previously impaired right elbow extensor motor function, achieved with a frequency of 2Hz, a pulse width of 550 ms, and an intensity of 5 amps. The patient reported a 60% improvement in spasticity and complete alleviation of pain, rating it at 0 out of 10. The patient approved the reporting of this case and results.

## DISCUSSION

Spinal cord injury can have devastating consequences for patients, affecting their physical, psychological and social well-being[11]. Common impairments associated with SCI are loss of motor and sensory function, bowel and bladder dysfunction, recurrent infections, autonomic dysreflexia, spasticity, contractures, and chronic pain. These impairments can also impose a significant financial burden on patients, who may incur costs ranging in the millions over their lifetime for SCI-related care [12]. The main goal of SCI treatment is to help patients enhance their functional abilities, mitigate pain, and ultimately cope with their condition in the best way possible.

Current treatment strategies for post-SCI spasticity involve multimodal strategies and a multidisciplinary approach. Initial treatment is typically conservative with passive muscle stretching and physical therapy, pharmacologic agents (tizanidine, benzodiazepines), onabotulinumtoxinA injections, or even surgical interventions (e.g., dorsal rhizotomy). Baclofen can additionally be administered through an intrathecal baclofen pump (ITB), providing sustained bolus release of the medication for spasticity management. While these treatment options are widely used, they come with limitations, including undesirable adverse effects, treatment resistance, and inconsistent results.

Technological advances have allowed for the development of minimally invasive techniques that can advance patient care and expand available treatment options. In 1967, by way of the "Gate Control Theory", it was postulated that the introduction of an exogenous electrical signal can potentially modulate the endogenous pain signals that coalesce within the dorsal column. Even though the exact underlying process of how SCS enhances functional recovery remains somewhat unclear at this time, the prevailing hypothesis is that the constant stimulation of afferent fibers in the dorsal root elevates the overall excitability of spinal circuits, making interneurons and motor neurons closer to their firing threshold and physiological state [13]. These effects enable these neurons to respond more effectively to the diminished inputs after an injury, thus increasing synaptic strength and plasticity. Although the mechanism is not fully understood, there is growing evidence that the suppression of motor neuron hyperactivity can also improve spasticity in patients with SCI [14]. Ongoing pathophysiologic research is needed to explain the underlying mechanism, but select patients are already experiencing tangible benefits from this treatment option.

SCS for improving spasticity after SCI is an emerging technology showing positive results overall [10, 15]. Although SCS is regularly studied in other uses, such as chronic pain, further literature is needed to continue the establishment of additional indications for the treatment modality. This case report illustrates the remarkable benefits of SCS for a patient with an incomplete AIS D SCI. After receiving SCS, he reported significant reductions in hand spasticity and neuropathic pain, as well as enhanced motor function and range of motion in his right arm. We hope that this case study helps provide evidence that spinal cord stimulators have the potential to positively impact the lives of patients with multiple ailments.

While there is emerging evidence of the potential role of SCS as a treatment modality for UMN-lesion-induced spasticity, more research is needed to assess efficacy, optimal stimulation parameters, and proper patient selection. With the advent of new technologies that can measure neural feedback during stimulation and the supraspinal/cortical changes occurring in addition to analgesia, there may be a lot more to be uncovered with future investigations.

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# Figure Legend(s):

Figure 1. Cervical spinal leads, AP Radiograph View. Leads placed in the C4-C5 vertebral levels. Figure 2. Thoracic spinal leads, AP Radiograph View. Leads placed midline at the level of the T11 vertebral body. Figure 3. Thoracic spinal leads, Lateral Radiograph View. Leads placed midline at the level of the T11 vertebral body.

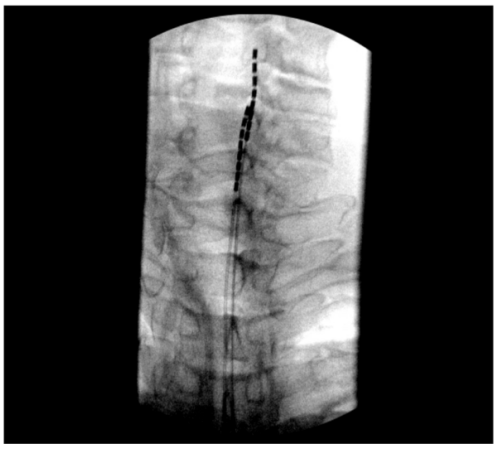

*Figure 1. Cervical spinal leads, AP Radiograph View*

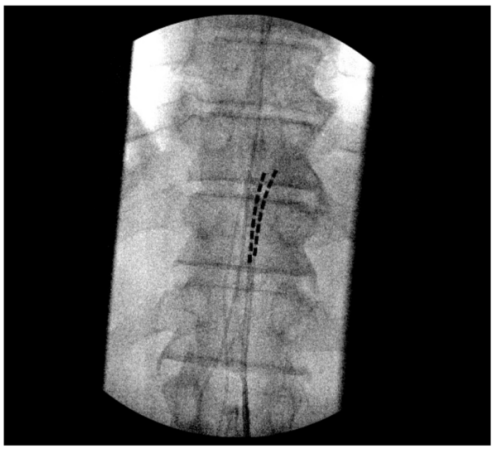

*Figure 2. Thoracic spinal leads, AP Radiograph View*

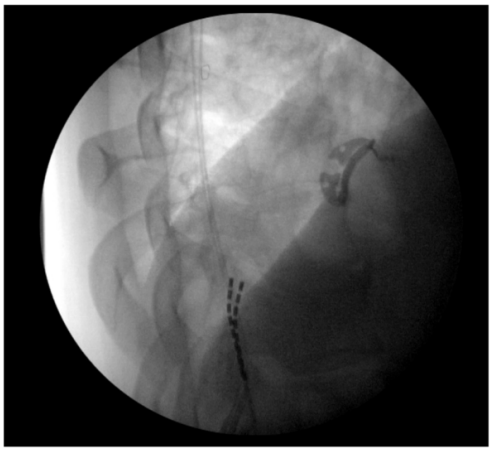

*Figure 3. Thoracic spinal leads, Lateral Radiograph View*